IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF NORTH CAROLINA
SOUTHERN DIVISION
No. 7:23-CV-897

IN RE:

CAMP LEJEUNE WATER LITIGATION

This Document Relates To:

***Amsler v. United States*, No. 7:23-cv-284**
***Connard v. United States*, No. 7:23-cv-1557**

UNITED STATES' MEMORANDUM OF LAW IN SUPPORT OF ITS MOTION TO EXCLUDE THE SPECIFIC CAUSATION OPINIONS OF DR. LUKASZ GONDEK

(LEUKEMIA)

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INTRODUCTION

Karen Marie Amsler, who lived with her family at Camp Lejeune in the 1960s, was diagnosed with Acute Lymphoblastic Leukemia ("ALL") in September 2020, when she was sixty years old. Stephen Matthew Connard, who was stationed at Camp Lejeune between 1977 and 1981, was diagnosed with Acute Myeloid Leukemia ("AML") in March 2001, at the age of forty-one. Both Ms. Amsler and Mr. Connard (through his estate representative) seek to recover damages under the Camp Lejeune Justice Act of 2022 ("CLJA").

To succeed on their claims, Ms. Amsler and Mr. Connard must demonstrate both that their leukemias "can be caused by the exposure to a degree of scientific certainty (general causation)," and that the "exposure was a cause in fact of his or her harm (specific causation)." *In re Camp Lejeune Water Litig.*, 736 F. Supp. 3d 311, 319 (E.D.N.C. 2024). As discussed below, Dr. Gondek's opinions should be excluded because they fail to meet the Federal Rule of Evidence 702 threshold of reliability under *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993). Specifically, although Dr. Gondek claims to have conducted a "differential etiology analysis" to reach his opinions, he failed to consider important factors that cut against his conclusions, namely, the long latency periods between Ms. Amsler's and Mr. Connard's exposure and diagnoses, as well as the likelihood that their leukemias were idiopathic or simply occurred by chance. This failure renders Dr. Gondek's opinions unreliable and therefore inadmissible.

BACKGROUND

I. Dr. Gondek's Methodology

On February 7, 2025, Dr. Lukasz Gondek proffered opinions that Ms. Amsler's ALL and Mr. Connard's AML were "more likely than not" caused by their exposure to contaminated water at Camp Lejeune. *See* Gondek SC Rep. (Amsler) at 12 (JA Ex. 455, D.E. 496-7), Gondek SC Rep. (Connard) at 15 (JA Ex. 456, D.E. 496-8). Dr. Gondek stated that, "[t]o establish causation," he

"rel[ied] upon a differential etiology analysis," which he defined as "a systematic method to evaluate all plausible causes of the disease" that "involves a consideration of potential confounders given the unique circumstances of the individual claimant involved." *Id.* at 6. He used his "years of education, training and experience to determine which factors can be ruled out as potential causes," contextualizing "insights from epidemiological studies and toxicological data . . . within the patient's individual exposure history, dose-response relationship, and latency period." *Id.* He stated that once he "synthesiz[es] these elements, [he] can provide a scientific opinion on whether the exposure to TCE, PCE and benzene are at least as likely as not to be the cause of the patient's condition." *Id.* He explained that if he could "say that a single factor is the most likely cause of [the individual's] leukemia, [his] causation opinion may be phrased in a 'more likely than not' manner," but if he could not "determine which is the most likely cause" due to "competing causes," his "causation opinion will be phrased in an 'at least as likely as not' manner." *Id.* Dr. Gondek's methodology does not provide for the possibility that the individual's leukemia may not have been caused by *any* of the "plausible causes" that he evaluated in his differential etiology analysis. Nor does his methodology account for the possibility that one or more of the "plausible causes," either singly or together, could outweigh exposure to contaminated water at Camp Lejeune as the cause of the leukemia. In either of those scenarios, one would not be able to conclude that exposure to contaminated water at Camp Lejeune is "at least as likely as not" the cause. *Id.*

II. Dr. Gondek Disregarded the Short Latency Period and Idiopathic Nature of Ms. Amsler's Specific Type of ALL.

Regarding Ms. Amsler, Dr. Gondek considered, and ruled out, the following potential risk factors or causes of Ms. Amsler's ALL: (1) Ms. Amsler's family history of cancer, (2) Ms. Amsler's social and medical history, and (3) Ms. Amsler's prior occupation as a microbiologist where she was exposed to various "biological hazardous material." Gondek SC Rep. (Amsler) at

8 (JA Ex. 455, D.E. 496-7).[1] This left only Ms. Amsler's "environmental exposure to benzene and TCE," *id.*,[2] which Dr. Gondek previously opined can cause leukemia, *see generally* Gondek GC Rep. (JA Ex. 96, D.E. 465-1). Accordingly, Dr. Gondek concluded that Ms. Amsler's "cumulative exposure" to TCE and benzene in the 1960s was "more likely than not the cause of her ALL" diagnosed in 2020. Gondek SC Rep. (Amsler) at 11-12 (JA Ex. 455, D.E. 496-7). Nowhere in his report did he discuss "the significance of Ms. Amsler's 53-year latency period from her last exposure at Camp Lejeune to her ALL diagnosis," Gondek SC Dep. Tr. at 209:2-8 (JA Ex. 603, D.E. 509-1), given the known "short latency period" between exposure to cytotoxic agents and acute leukemia with KMT2A/11q23 rearrangements,[3] *id.* at 189:1-20.[4]

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[1] In his report, Dr. Gondek considered the "molecular and cytogenetic profile" of her specific type of ALL, stating that 11q23 (KMT2A), "is more likely to be associated with exposures to cytotoxic agents such as benzene, TCE and PCE." Gondek SC Rep. (Amsler) at 7 (JA Ex. 455, D.E. 496-7). However, he expressly retracted this statement at his deposition, acknowledging that he should have said "chromosomal rearrangements" instead of 11q23 rearrangements because, as United States expert, Dr. Erba, pointed out in his rebuttal report, the "particular rearrangement 11q23 was not observed" in individuals exposed to benzene, TCE, and PCE. Gondek SC Dep. Tr. at 81:18-82:16 (JA Ex. 603, D.E. 509-1).

[2] Despite his claim that he considered evidence specific to the individual's "dose-response relationship" and "latency period," Gondek SC Rep. (Amsler) at 6 (JA Ex. 455, D.E. 496-7), Dr. Gondek's report contains no discussion of either consideration specific to Ms. Amsler. Also, despite his testimony that "age is always a risk factor for most of the malignancies," Gondek SC Dep. Tr. at 206:20-22 (JA Ex. 603, D.E. 509-1), and perhaps even "the most significant factor, because the longer we'll live, the more mutations we accumulate," *id.* at 179:2-8, Dr. Gondek's report failed to acknowledge or discuss the significance of Ms. Amsler's advancing age when she was diagnosed with ALL.

[3] Dr. Gondek explained that KMT2A "stands for lysine methyltransferase"—"K is lysine, and MT is methyltransferase," and "2A is a subtype of the enzyme." Gondek SC Dep. Tr. at 180:20-181:7 (JA Ex. 603, D.E. 509-1). KMT2A "is the gene that encodes this enzyme, and it's located on chromosome 11q23." *Id.* at 181:6-7. "[S]ometimes this gene, KMT2A, gets rearranged to a different part of . . . the genome, and . . . it may be attached to other chromosomes," causing "abnormal activity of this gene KMT2A." *Id.* at 181:8-17.

[4] On April 8, 2025, the United States proffered alternate explanations for Ms. Amsler's ALL through its leukemia expert, Dr. Harry Erba, who observed that "over 90% of [ALL] cases are idiopathic without an obvious etiology." Erba Rep. (Amsler) at 5 (JA Ex. 473, D.E. 497-6). Dr. Erba further opined that, based on the "very short latency between development of a KMT2A

At his deposition, Dr. Gondek acknowledged that ALL with KMT2A/11q23 rearrangement, the specific genetic mutation that presented with Ms. Amsler's ALL, usually "aris[es] within five years of treatment with chemotherapeutic agents." Gondek SC Dep. Tr. at 184:5-17 (JA Ex. 603, D.E. 509-1). He also admitted that the scientific literature he cited in his report consistently describes a "short latency period" of less than three years between exposure to cytotoxic agents and acute leukemia with 11q23 rearrangements. *Id.* at 189:1-20. He provided no support whatsoever for his opinion that Ms. Amsler's ALL 11q23 was caused by her exposure to Camp Lejeune water more than fifty years before her diagnosis, other than "we knew Ms. Amsler was exposed to – to cytotoxic agents." *Id.* at 205:16-19.

Dr. Gondek also agreed that a "majority of [ALL] cases" are "idiopathic," which he defined this way:

> So idiopathic is used when we don't know what caused cancer. So what it means is, there is a lot of things we know, and there is probably more that we don't know. So what idiopathic means is that by looking at all the possible causes, we didn't find anything, and there is no smoking guns. But it doesn't necessarily mean that someone's cancer was not induced by external factors. We just don't know about them. And that's what idiopathic means. We don't know what caused the cancer. It doesn't necessarily mean that there was nothing there that caused the cancer.

Id. at 204:19-205:8. When asked why he did not address idiopathy in his report, he replied, "since we have a cause here, which is exposure to cytotoxic agents, I don't think we can discuss idiopathic causes. Like I took into consideration idiopathic, meaning unknown, but in [Ms. Amsler's] case we have a known cause, which is exposure to benzene and TCE." *Id.* at 210:19-211:7.

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rearrangement and development of acute leukemia," it "is highly improbable, bordering on impossible, that any possible chemical exposure more than five decades earlier could be a direct cause of Ms. Amsler's specific subtype of ALL with KMT2A rearrangement." *Id.* at 14, 15. Dr. Gondek did not proffer a rebuttal to Dr. Erba's opinions.

III. Dr. Gondek Also Ignored the Latency Period and Idiopathic Nature of Mr. Connard's AML.

As for Mr. Connard, Dr. Gondek considered the following factors: (1) Mr. Connard's specific type of genetic rearrangement (monosomy 7) accompanying his AML diagnosis,[5] (2) Mr. Connard's family's medical history, (3) Mr. Connard's social and medical history, and (4) Mr. Connard's occupational exposure to potential carcinogens, including at Camp Lejeune.), Gondek SC Rep. (Connard) at 6-9 (JA Ex. 456, D.E. 496-8). Finding no "hereditary disposition," "underlying medical conditions, or history of radiation or chemotherapy prior to [Mr. Connard's] diagnosis of AML," *id.* at 14, Dr. Gondek "conclude[d] that Mr. Connard's exposure to TCE, PCE and benzene through contaminated water at Camp Lejeune was more likely than not the cause of his AML and the complications that led to his death," *id.* at 15. Like his report for Ms. Amsler, Dr. Gondek's report for Mr. Connard makes no mention of the near-twenty-year latency period between Mr. Connard's time at Camp Lejeune and his AML diagnosis, or the highly idiopathic or sporadic nature of AML.[6]

At his deposition, Dr. Gondek admitted that "[i]n most cases, we can't pinpoint to a cause" of AML, Gondek SC Dep. Tr. at 280:7-16 (JA Ex. 603, D.E. 509-1), and that Mr. Connard's AML

[5] Dr. Gondek stated in his report that Mr. Connard's monosomy 7 "further support[ed] [his] opinion" because of studies that "demonstrated the presence of this abnormalities in blood cells collected from humans exposed to benzene." Gondek SC Rep. (Connard) at 7 (JA Ex. 456, D.E. 496-8). However, he later admitted at his deposition that "monosomy 7 . . . can be present in people that [] don't have clear evidence of exposure. Doesn't necessarily mean they were not exposed; we just don't know of it." Gondek SC Dep. Tr. at 275:21-276:5 (JA Ex. 603, D.E. 509-1).

[6] United States' expert, Dr. Erba, also proffered alternate explanations for Mr. Connard's AML, observing that (1) "most AML cases (85-90%) are idiopathic;" and (2) "AML due to prior exposure to DNA damaging agents such as alkylating chemotherapy and ionizing radiation typically occurs within 10 years of exposure." Erba Rep. (Connard) at 6 (JA Ex. 474, D.E. 497-7) (discussing "a series of 65 patients exposed to alkylating agent chemotherapy and/or radiation therapy" where the latency period "ranged from 11 to 192 months [16 years] with a median of 58 months" and "only 6 of 65 cases (14%) had a latency over 120 months (10 years)"). *Id.* Dr. Gondek did not provide a rebuttal report responding to Dr. Erba's opinions.

could have been "idiopathic" or "sporadic," *id.* at 287:3-16.[7] He also admitted that there are "a lot of papers showing that the mutations [causing AML] happen spontaneously, and there is a rate of spontaneous mutation." *Id.* at 282:6-10. Even then, he could not explain why he concluded that Mr. Connard's AML was *not* idiopathic or sporadic, despite his admission that, "[i]f Mr. Connard had never been to Camp Lejeune," he "would not be able to identify the cause of his malignancy" and "would assume that the [AML] was idiopathic." *Id.* at 288:21-289:8.

Dr. Gondek also acknowledged that the latency period between exposure to known chemotherapeutic agents and an AML diagnosis is between two and ten years. *See id.* at 248:20-249:7 (stating that, "in AML, the latency is between two and three, up to five years for topoisomerase II inhibitors," and for "alkylating agents . . . probably closer to eight to ten years."). His general causation report discussed the "Linet et al. 2019" study, which "found increased MDS/AML particularly in younger patients before age 30 years within 2-10 years of benzene exposure." Gondek GC Rep. at 15 (JA Ex. 96, D.E. 465-1). At his general causation deposition, Dr. Gondek acknowledged that the *Linet* study found "little evidence of exposure response after at least ten years regardless of age at first exposure;" Gondek GC Dep. Tr. at 167:20-168:3 (JA Ex. 160, D.E. 469-14), consequently, he admitted that the *Linet* study "supports a ten-year etiological window," *id.* at 168:4-18.[8] Dr. Gondek also acknowledged another study cited in his report, authored by Glass et al. in 2006, which "showed that the risk of leukemia is associated with

[7] According to Dr. Gondek, "idiopathic means that we don't know the etiology of the cancer, but it doesn't mean that one doesn't exist. We just don't know about it. When we are talking about sporadic cancer, we're kind of implying that the cancer was just by chance." Gondek SC Dep. Tr. at 287:5-12 (JA Ex. 603, D.E. 509-1).

[8] *See* **Exhibit 1**, Martha S. Linet et al., *Benzene Exposure Response and Risk of Myeloid Neoplasms in Chinese Workers: A Multicenter Case–Cohort Study*, 111 J. NAT'L CANCER INST. 465, 465 (2019).

exposure within 15 years of diagnosis,"[9] and that the association of leukemia to an exposure more than fifteen years earlier "is weak." *Id.* at 169:5-15.[10] Despite this, Dr. Gondek "determined that the water at the Camp Lejeune was the only risk factor that [he] could not rule out" for Mr. Connard, *id.* at 288:13-20, while admitting that for the "majority of cases [of AML], we can't really pinpoint the cause of the disease" and "people can get AML just by chance," Gondek SC Dep. Tr. at 281:21-282:10 (JA Ex. 603, D.E. 509-1).

LEGAL STANDARD

An expert's testimony is admissible if "it rests on a reliable foundation and is relevant to the task at hand." *Belville v. Ford Motor Co.*, 919 F.3d 224, 232 (4th Cir. 2019) (citing *Daubert*, 509 U.S. at 597). "The trial court must perform the special gatekeeping obligation of ensuring that expert testimony meets both requirements." *Eshelman v. Puma Biotechnology, Inc.*, No. 7:16-CV-18-D, 2019 WL 1092572, at *3 (E.D.N.C. Mar. 8, 2019) (Dever, J.) (citing *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 147 (1999)). Expert testimony is admissible only if it (a) "will help the trier of fact to understand the evidence or to determine a fact in issue," (b) "is based upon sufficient facts or data," (c) "is the product of reliable principles and methods," and (d) "reflects a reliable application of the principles and methods to the facts of the case." Fed. R. Evid. 702.

[9] *See* **Exhibit 2**, Deborah C. Glass et al., *The HealthWatch Case–Control Study of Leukemia and Benzene: The Story So Far*, 1076 ANN. N.Y. ACAD. SCI. 80, 85 (2006).

[10] At his specific causation deposition, Dr. Gondek claimed that "there are studies . . . that actually show the increased incidence of leukemia even 30 years after exposure," and that he thought he "cited some of the studies in his report in General Causation." Gondek SC Dep. Tr. at 312:13-21 (JA Ex. 603, D.E. 509-1). However, his general causation report does not include any discussion of latency other than the *Linet* study referenced above, and the "Rinsky" study he referred to at his deposition, *id.*, defined latency as "the length of time (in years) from the date of first exposure until death," not leukemia incidence, as Dr. Gondek claimed. *See* **Exhibit 3**, Robert A. Rinsky et al., *Benzene and Leukemia: An Epidemiological Assessment*, 316 NEW ENG. J. MED. 1044, 1047 (1987). Moreover, the *Rinsky* study found that "[n]o apparent pattern was evident for these *deaths* with regard to latency, which ranged from under 5 to over 30 years; however, seven of the nine persons with leukemia had less than 20 years of latency." *Id.* at 1046 (emphasis added).

Differential etiology is "a standard scientific technique of identifying the cause of a medical problem by eliminating the likely causes until the most probable one is isolated." *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 262 (4th Cir. 1999).[11] The process is straightforward: The expert begins by "determining the possible causes for the patient's symptoms and then eliminat[es] each of these potential causes until reaching one that cannot be ruled out or determining which of those that cannot be excluded is the most likely." *Id.* at 262 (citation omitted). A *reliable* differential etiology can be used to show specific causation. *See, e.g.*, *id*.

"[N]ot every opinion that is reached via a differential diagnosis method will meet the standard of reliability required by *Daubert*." *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs., & Prods. Liab. Litig.*, 892 F.3d 624, 643 (4th Cir. 2018) (quotation omitted)). To satisfy *Daubert* and Rule 702, the expert must "take serious account" of other potential causes for the disease and reliably rule them out. *Westberry*, 178 F.3d at 265. "[A]n expert does not establish the reliability of his techniques or the validity of his conclusions simply by claiming that he performed a differential diagnosis on a patient." *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1253 (11th Cir. 2005). While an expert "need not rule out all possible alternative causes," he "must at least consider" other potential causes. *In re Lipitor*, 892 F.3d at 643 (citation and quotation omitted)); *see also Nix v. Chemours Co. FC*, No. 7:17-cv-189, 2023 WL 6471690, at *8 (E.D.N.C. Oct. 4, 2023) (Dever, J.) ("When there are multiple avenues of potential causation, an expert must account

[11] Although some cases have used the terms "differential etiology" and "differential diagnosis" interchangeably, they refer to separate processes. *See, e.g.*, *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs., & Prods. Liab. Litig.*, 150 F. Supp. 3d 644, 660 n.17 (D.S.C. 2015). "Differential diagnosis" refers to a process for "identifying a set of diseases or illnesses responsible for [a] patient's symptoms," and "differential etiology" refers to a process for "identifying the causal factors involved in an individual's disease or illness." Fed. Jud. Ctr., *Reference Manual on Scientific Evidence* 617 n.211 (3d ed. 2011) [hereinafter *RMSE*]. Because the latter method is "more accurately referred to as differential etiology," the United States uses that term. *Id.* at 617.

for alternative causes."); *Eshelman*, 2019 WL 1092572, at *6 (finding expert opinion unreliable, in part, because the expert "also fail[ed] to adequately consider alternate explanations" (citing *Claar v. Burlington N. R.R.*, 29 F.3d 499, 502 (9th Cir. 1994)).

"A differential diagnosis that fails to take serious account of other potential causes may be so lacking that it cannot provide a reliable basis for an opinion on causation." *Westberry*, 178 F.3d at 265. As the Fourth Circuit has made clear, if an expert "utterly fails to consider alternative causes" or offers "*no* explanation for why she has concluded an [alternative cause] was not the sole cause," exclusion is warranted. *Id.* at 265-66 (emphasis in original); *see also Cooper v. Smith & Nephew, Inc.*, 259 F.3d 194, 202 (4th Cir. 2001).

ARGUMENT

Although Dr. Gondek claims that he conducted a differential etiology analysis to establish specific causation as to Ms. Amsler and Mr. Connard, he ignored significant factors related to their leukemias that directly contradicted his opinions. His failure to adequately "account for alternative causes," *Nix*, 2023 WL 6471690, at *8, renders his analyses unreliable and warrants exclusion of his opinions.

I. Dr. Gondek Failed to Consider the Latency Periods Between Exposure and Diagnosis.

Dr. Gondek stated in his specific causation reports that his "differential etiology analysis" includes an evaluation of "all plausible causes of the disease." Gondek SC Rep. (Amsler) at 6 (JA Ex. 455, D.E. 496-7), Gondek SC Rep. (Connard) at 6 (JA Ex. 456, D.E. 496-8). Part of his "systematic method" is to "contextualize[]" the scientific data with "the patient's individual exposure history, dose-response relationship, and *latency period*." *Id.* (emphasis added). However, Dr. Gondek did not even mention the latency period in either of his reports, much less "synthesiz[e]" it in order to provide his "scientific opinion" on specific causation. *Id.* In fact, Dr. Gondek did not even submit a rebuttal or supplemental report explaining a consideration of latency

for these cases after Dr. Erba pointed out all of the scientific support for a "very short latency" for ALL with KMT2A arrangements, Erba Rep. (Amsler) at 16 (JA Ex. 473, D.E. 497-6), and the latency period of less than fifteen years for AML, Erba Rep. (Connard) at 12, 15 (JA Ex. 474, D.E. 497-7). The short latency periods *alone* contradict (if not completely negate) the likelihood that either Ms. Amsler's or Mr. Connard's leukemia was caused by exposure to Camp Lejeune water decades prior to their diagnoses.

With respect to Ms. Amsler, Dr. Gondek testified at his deposition that ALL 11q23 usually "aris[es] within five years" of exposure to cytotoxic agents such as chemotherapy, Gondek SC Dep. Tr. at 184:15-17 (JA Ex. 603, D.E. 509-1), and acknowledged that several studies he cited in his report describe the latency period as "within two to three years" after exposure, *id.* at 186:4-189:15. Despite this short latency period described in the scientific literature, Dr. Gondek admittedly "did not discuss the significance to Ms. Amsler's 53-year latency period from her last exposure at Camp Lejeune to her ALL diagnosis." *Id.* at 209:2-8. By offering "*no* explanation" as to how Ms. Amsler could have developed ALL 11q23 ***more than five decades*** after toxic exposure—when the scientific literature consistently recognizes a ***two- to three-year*** latency period—Dr. Gondek failed to reliably demonstrate that her exposure to Camp Lejeune water was "the sole cause" of her ALL. *Westberry*, 178 F.3d at 265-66.

With respect to Mr. Connard, Dr. Gondek likewise failed to explain—or point to any relevant scientific literature explaining—why "the water at the Camp Lejeune," to which Mr. Connard was last exposed twenty years before his AML diagnosis, "was the only risk factor that [he] could not rule out." Gondek SC Dep. Tr. at 288:13-20 (JA Ex. 603, D.E. 509-1), even though he agreed that the latency period between exposure to toxic chemotherapy and AML is between two and ten years. *Id.* at 248:20-249:7. At his general causation deposition, Dr. Gondek

acknowledged that the studies cited in his report consistently described a latency period of less than fifteen years for AML. *See* Gondek GC Dep. Tr. at 167:20-169:15 (JA Ex. 160, D.E. 469-14). Dr. Gondek may seek to rely on the *Rinsky* study to support a longer latency period. But any reliance on that study for a thirty-plus year latency period is misplaced. The study did not analyze the latency period between exposure and leukemia *diagnosis*—it analyzed the latency period between exposure and *death* from leukemia. *See* Ex. 3, Robert A. Rinsky et al., *Benzene and Leukemia: An Epidemiological Assessment*, 316 NEW ENG. J. MED. 1044, 1047 (1987). Moreover, the *Rinsky* study expressly found "no apparent pattern" for the "deaths with regard to latency." *Id.* at 1046. In short, the scientific literature on AML, upon which experts for both Plaintiffs and the United States rely for their general causation opinions, concludes that AML occurs within fifteen years of toxic exposure. After fifteen years, the risk of such exposure causing AML is significantly reduced, if not altogether eliminated.

In sum, Dr. Gondek failed to "synthesize" into his "differential etiology analysis" the significant latency periods between both Ms. Amsler's and Mr. Connard's exposure and diagnoses, Gondek SC Rep. (Amsler) at 6 (JA Ex. 455, D.E. 496-7); Gondek SC Rep. (Connard) at 6 (JA Ex. 456, D.E. 496-8), and failed to provide a "reasonable explanation" for his failure to rule out exposure to Camp Lejeune water as a plausible cause of their leukemias, *Lightfoot*, 2018 WL 4517616, at *22. Consequently, Dr. Gondek's opinions are unreliable and this Court is justified in excluding his testimony in both cases. *See Westberry*, 178 F.3d at 265-66; *Cooper*, 259 F.3d at 202.

II. Dr. Gondek Failed to Consider the Likelihood that Ms. Amsler's and Mr. Connard's Leukemias had an Unknown Cause or Simply Occurred by Chance.

In addition to Dr. Gondek's failure to consider the latency periods between Ms. Amsler's and Mr. Connard's exposures and diagnoses, Dr. Gondek failed to discuss the idiopathic nature of

leukemias, including ALL and AML. As Dr. Gondek admitted at his deposition, a "majority of [ALL] cases" are "idiopathic," meaning "we don't know what caused it." Gondek SC Dep. Tr. at 205:9-206:2 (JA Ex. 603, D.E. 509-1). He also admitted that, "in most cases, we can't pinpoint to a cause" of AML, *id.* at 280:7-16, and there are "a lot of papers showing that the mutations [causing AML] happen spontaneously, and there is a rate of spontaneous mutation," *id.* at 282:6-10. Despite the wealth of scientific literature on leukemia and idiopathy, Dr. Gondek failed to mention the word "idiopathic" or "idiopathy" in either of his specific causation reports. *See* Gondek SC Rep. (Amsler) (JA Ex. 455, D.E. 496-7), *passim*; Gondek SC Rep. (Connard) (JA Ex. 456, D.E. 496-8), *passim.* At deposition, his circular explanation for this glaring omission was that "we have a cause here, which is exposure to cytotoxic agents," Gondek SC Dep. Tr. at 210:19-211:7 (JA Ex. 603, D.E. 509-1). The Fourth Circuit has consistently cautioned against this type of backward reasoning. *See In re Lipitor*, 892 F.3d at 644-45 (affirming exclusion of expert testimony where the expert "appeared to simply conclude that so long as the patient took Lipitor and developed diabetes, then Lipitor was a substantial contributing factor") (citations and quotation marks omitted); *Westberry*, 178 F.3d at 265 (advising that "the mere fact that two events correspond in time does not mean that the two necessarily are related in any causative fashion").[12] "Simply put, Daubert requires more." *In re Lipitor*, 892 F.3d at 645.

[12] Dr. Gondek also admitted that he "was not aware that there are no actual samples taken from the time that Ms. Amsler was at Camp Lejeune," and that Ms. Amsler's exposure estimates were "calculated on some sort of modeling." Gondek SC Dep. Tr. at 211:21-212:7 (JA Ex. 603, D.E. 509-1). As the United States has previously argued in this litigation, the "ATSDR's water models are unreliable, scientifically invalid, and not sufficiently accurate for the purpose of determining absolute concentration levels over particular time periods for individual exposure determinations," in large part, because they "relied on the limited contaminant concentration sampling data that was available from the early to mid-1980s to simulate estimated contaminant concentration levels in drinking water at Camp Lejeune for more than 30 years into the past." D.E. 368 at 1-2.

When ruling in diagnoses for the differential, the expert must do more than identify "risk factors," and the expert "cannot merely conclude that all risk factors" are causes. *Guinn v. AstraZeneca Pharm. LP*, 602 F.3d 1245, 1255 (11th Cir. 2010). After all, "risk factor" and "cause" are not synonymous. *See, e.g.*, *id.* ("The fact that exposure to [a substance] may be a risk factor for [a disease] does not make it an actual cause simply because [the disease] developed." (citation and quotation omitted)). Many patients have risk factors and not the disease, and many patients have the disease with no risk factors. *See, e.g.*, *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs., & Prods. Liab. Litig.*, 150 F. Supp. 3d 644, 657 (D.S.C. 2015) (describing patients with diabetes risk factors that do not have diabetes); *see also, e.g.*, Gondek SC Dep. Tr. at 177:4-179:21 (JA Ex. 603, D.E. 509-1) (agreeing that "[m]ost people who get acute lymphocytic leukemia have no known risk factors, so there is no way to prevent these leukemias from developing").

While some diseases have well-established causes (e.g., smoking and lung cancer), many diseases present without identifiable causes and are "idiopathic." When there are many idiopathic cases of a particular disease, it is "impossible to ignore and difficult to rule out" the possibility of idiopathy. *Tamraz v. Lincoln Elec. Co.*, 620 F.3d 665, 675 (6th Cir. 2010). The reason for this is, in part, mathematical. *See*, *e.g.*, *Whiting v. Bos. Edison Co.*, 891 F. Supp. 12, 21 n.41 (D. Mass. 1995) ("If 90 percent of the causes of a disease are unknown, it is impossible to eliminate an unknown disease as the efficient cause of a patient's illness."). Indeed, "for diseases for which the causes are largely unknown . . . a differential etiology is of little benefit." FED. JUDICIAL CTR., REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 618 (3d ed. 2011). Accordingly, the differential etiology methodology becomes less reliable as the potential number of idiopathic cases increases, and this is particularly so where the majority of cases are likely idiopathic (as, for example, for ALL and AML). *See, e.g.*, *Hall v. Conoco Inc.*, 886 F.3d 1308, 1315 (10th Cir. 2018) ("Because

idiopathy accounts for more than half of the cases of [the disease at issue], a differential diagnosis could be considered inherently unreliable here."); *Milward v. Rust-Oleum Corp.*, 820 F.3d 469, 476 (1st Cir. 2016) (affirming exclusion of expert because "the extraordinary number of idiopathic . . . cases, coupled with the lack of a reliable means to rule out an idiopathic diagnosis here, muted [the expert's] ability to reliably apply" the differential diagnosis method); *Bland v. Verizon Wireless, (VAW) L.L.C.*, 538 F.3d 893, 897 (8th Cir. 2008) ("Where the cause of the condition is unknown in the majority of cases, [an expert] cannot properly conclude, based upon a differential diagnosis, [that the patient's exposure] was 'the most probable cause' of [the disease].").

In this litigation, Plaintiffs' burden under the CLJA is to show that the water supplied at Camp Lejeune is "at least as likely as not" the specific cause of their injuries. Just as the expert must weigh the Camp Lejeune-related risks against the background risk, the expert must also weigh the Camp Lejeune-related risks against the risks attributable to other, unexcluded causes (like smoking). Where the Camp Lejeune-related risks are only a fraction of the plaintiff's total disease-risk, it is difficult to draw any reliable conclusions concerning but-for causation as to the Camp Lejeune water even under an "as likely as not" burden of proof. *See United States v. Alvarado*, 816 F.3d 242, 249 (4th Cir. 2016) (holding but-for causation was not established where exposure to toxin "plays merely a nonessential contributing role in [the person's] death").

Here, Dr. Gondek conceded that ALL and AML are largely idiopathic diseases. *See* Gondek SC Dep. Tr. at 205:9-206:2 (JA Ex. 603, D.E. 509-1) ("I would agree that most often we don't know what caused [ALL]."); *id.* at 280:14-18 ("In most cases [of AML], we can't pinpoint to a cause."); *id.* at 282:3-17 (agreeing "that people can get AML just by chance," and acknowledging "a lot of papers showing that the mutations can happen spontaneously"). But he

failed to consider idiopathic causation at all. His reports do not analyze, discuss—or even refer to—idiopathy or anything similar. *See generally* Gondek SC Rep. (Amsler) (JA Ex. 455, D.E. 496-7); Gondek SC Rep. (Connard) (JA Ex. 456, D.E. 496-8). That omission justifies exclusion. *See Hall*, 886 F.3d at 1314-16 (affirming exclusion of expert for failing to rule out idiopathy in a case involving benzene and acute myeloid leukemia in light of undisputed testimony that 70-75% of AML cases are idiopathic); *Milward*, 820 F.3d at 475-76 (affirming exclusion of expert who ruled out idiopathic causation of acute promyelocytic leukemia by ruling in benzene as a cause because such reasoning was "circular" and of limited utility when 70-80% of APL cases are idiopathic); *Chapman*, 766 F.3d at 1311; *Kilpatrick*, 613 F.3d at 1343 (affirming exclusion of expert who admitted he could not explain why "potentially unknown, or idiopathic alternative causes were not ruled out").[13] At his deposition, although Dr. Gondek claimed that he does consider idiopathy in his differentials, he testified that he did not consider idiopathic causation for Ms. Amsler or Mr. Connard solely because he had identified their Camp Lejeune exposure as a possible cause.[14] In other words, once Dr. Gondek concluded that exposure to Camp Lejeune water was a "competing

[13] In *Lightfoot v. Georgia-Pacific Wood Prods., LLC*, the defendants argued that two specific causation experts' opinions were unreliable because they failed to rule out idiopathic cause for a form of sinonasal cancer allegedly caused by wood dust exposure, but Judge Flanagan disagreed. *See* 2018 WL 4517616, at *21-22 (E.D.N.C. Sept. 20, 2018). However, because the experts did, in fact, "explain why they ruled out the possibility that plaintiff's [cancer] was idiopathic under the circumstances of [that] case[,]" *id.* at *21, *Lightfoot* is distinguishable on its face. To the extent *Lightfoot* reasons that a specific causation opinion need not consider idiopathy to be reliable (and especially for cancers like ALL and AML that are idiopathic in most cases), the United States respectfully disagrees and submits that this Court should not follow such reasoning.

[14] *See, e.g.*, Gondek SC Dep. Tr. at 205:13-21 (JA Ex. 603, D.E. 509-1) (testifying that idiopathy was not "pertinent to [Ms. Amsler's] case, because . . . we knew [she] was exposed to – to cytotoxic agents"); 210:19-211:7 (testifying that "since we have a cause here, which is exposure to cytotoxic agents, I don't think we can discuss idiopathic causes. Like I took into consideration idiopathic, meaning unknown, but in her case we have a known cause, which is exposure to benzene and TCE"); 286:18-287:2 (testifying that he did not discuss idiopathic causation in Mr. Connard's case because "in that case, we obviously had offending agents").

cause" of Ms. Amsler's and Mr. Connard's leukemia, he considered his analysis complete, and concluded that the Camp Lejeune water was "at least as likely as not" the cause, without evaluating that cause against other "competing" factors such as latency, or other explanations such as idiopathy. Gondek SC Rep. (Amsler) at 12 (JA Ex. 455, D.E. 496-7), Gondek SC Rep. (Connard) at 15 (JA Ex. 456, D.E. 496-8). This is not a reliable differential diagnosis that takes "serious account" of the potential causes of disease. *See Westberry,* 178 F.3d at 265. *See also In re Lipitor*, 892 F.3d at 643-44. Dr. Gondek's failure to analyze or weigh idiopathy as an alternate risk factor—for diseases like ALL and AML that are usually idiopathic—renders his "differential etiology analysis" unreliable and inadmissible under Rule 702(d).

CONCLUSION

For the foregoing reasons, the United States respectfully requests that this Court exclude the specific causation testimony of Dr. Gondek with respect to Ms. Karen Amsler and Mr. Stephen Connard.

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CERTIFICATE OF SERVICE

I hereby certify that on September 9, 2025, I electronically filed the foregoing using the Court's Electronic Case Filing system, which will send notice to all counsel of record.

/s/ Jennifer E. Adams
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